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Cadmium Uptake from Feed and Its Distribution to Food Products of Livestock

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Distribution of cadmium (Cd) into the edible products of three species of food-producing animals was investigated during long-term dietary administration of supplemental cadmium chloride.

Cows were exposed to 0.2 (control), 2.4, and 11.3 ppm Cd on whole ration basis for a period of three months followed by a three-month period on control ration. No accumulation of Cd occurred in milk, muscle, or bone at any of the time periods. A dose-related increase of Cd was observed in liver and kidney. The Cd concentration in these organs showed a gradual rise even when the animals were given control ration for three months after an initial three-month exposure to Cd; this observation suggests a mobilization and redistribution of this metal from other tissues.

Dietary Cd levels in swine rations were 0.2, 2.4, and 10.1 ppm. The highest level of Cd produced a slightly reduced growth rate in swine. No accumulation of Cd was observed in muscle, bone, or brain. Liver and kidney showed a treatment and time-related increase in cadmium values at 6 and 12 weeks on experiment. During a three-month depletion phase after an initial three months of Cd administration, no further change in liver and kidney Cd levels was observed.

White Leghorn chickens were treated by administering 0.3, 1.9, and 13.1 ppm Cd in their diets for up to 6 months. No accumulation of Cd occurred in eggs or bones. A slight increase of Cd level was observed in chicken muscle after six months of exposure. Liver and kidney had the highest levels of Cd, which showed a dose and time-related increase in these organs. No depletion of liver and kidney Cd was seen during seven weeks following a six-week treatment period.

In all three species, the kidney Cd levels were severalfold higher than those of liver at all dietary levels. In swine, the renal cortex and medulla had similar Cd concentration in control animals but in all animals exposed to supplemental Cd, a dramatic rise in Cd levels in renal cortex was observed. Medullary Cd did not show a proportional time and dose-related increase in Cd levels, although the levels showed some increase. In kidney and liver of all three species the Cd levels showed a positive correlation with the amount of Cd-binding protein in these tissues. Induced levels of this metal-binding protein may explain accumulation and persistence of Cd-residues in these organs. In all three species, the concentrations of renal Cd-binding protein increased at a rate greater than those in liver.

Although the food-producing animals may act as an effective filter of Cd in the case of an environmental increase of this metal, consumption of visceral organs from such animals may pose a hazard. This is particularly critical since the daily intake of Cd in human diet is already estimated to exceed the tolerance limits suggested by WHO/FAO.

Introduction

Cadmium is a heavy metal uniformly distributed throughout the earth's crust. Although it has been observed almost in all living tissues, no biological role for its presence has been ascertained and it is always considered as an undesirable toxic element. Excess of cadmium is not compatible with normal physiological processes and toxicity due to accidental exposure to this element has been well known. Repeated dosing of animals with cadmium caused reduced erythrocyte ALAD activity and decreased survival times (*1*).

Cadmium has been found in air in concentrations of < 0.01 to $0.35\ \mu g/m^3$ (*2*), with concentrations usually highest in cities with a considerable industrial activity. Cadmium levels of most soils are

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below 1 ppm, perhaps about 0.4 ppm on the average (*3*). Soil obtained in the vicinity of smelters and near highways may contain considerably high amounts of cadmium (*4, 5*). Sewage sludge contains relatively high amounts of cadmium, the concentrations ranging from 3 to over 3000 ppm, with a mean value of 106 ppm (*6*). Although the accumulation of this metal in plants growing on soil rich in cadmium is not appreciable, the application of sewage sludge on the cropland has posed a hazard of accumulation of this element in the food chain.

Tissues and products from domestic animals are a vital part of human food. Meat, milk, eggs, and even other organs and tissues are largely used for human consumption. Bones and other organs not generally used by humans are often incorporated in other animal feeds as an important source of nutrients. Some of the metals are known to be cumulative in tissues like bone, liver, and kidneys. The epidemic of mercury poisoning in Japan (Minamata episode) through accumulated metal in fish has illustrated the possibility of increased uptake of toxic heavy metals by humans through the food chain, thus, obtaining information on the distribution of toxic metals in actual food-producing species of animals is very important. At the same time, it is also important that the exposure levels to these toxic metals in various species be practical, i.e., close to the metal contents that have been reported or may be found in contaminated animal feeds.

It is recognized that long-term studies in large animals are difficult and expensive, but the importance and significance of data that must be obtained, considering the importance of such information in human health, overrides the cost considerations. In the study presented in this report we have studied the effects and accumulation of cadmium in three domestic animal species that are important human food producers, i.e., dairy cows, growing swine, and laying chickens. Low level exposures to the toxic metal have been related to known clinical and toxic effects.

Methods

Description of Animals and Management

The three animal species used in cadmium feeding studies were two-year old cows, growing swine, and laying chickens. All animals were procured in a healthy condition and maintained on the premises for at least one month before the experiments were started. The cows were tested for brucellosis and tuberculosis. No disease problems were observed in any species of animals.

Twelve Holstein heifers, all in their first gestation, approximately 420 kg body weight were procured. The animals were obtained prior to their calving and maintained in open pastures until they calved. After calving they were housed in individual stalls. The stalls were approximately 1.15 × 2 in size and had a 0.9 m manger in front. All exposed metal portions on the stalls were painted with an epoxy (Tile-Clad) paint to avoid licking of metal or contamination of feed. The animals were occasionally exercised and were supported in the stalls on a rubber padding. The cows were watered manually three times a day (120 liters of water each day) and were offered 10-15 kg of hay and 2.8 kg of grain (16% protein dairy ration, given in two divided portions after each milking) per day. The amount of refused hay (if any) was periodically measured. Every afternoon, the animals were also given 100 g of soybean meal (containing the $CdCl_2$ daily dose) layered on the top of their grain. Preliminary trials showed that this was an effective way of introducing a uniform amount of Cd each day without any problems. The animals did not waste any amount of this soybean meal.

All animals were milked twice daily. The morning milking was usually between 6 and 8 a.m. and the afternoon between 4 and 6 p.m. Milk samples were saved for analyses and the milk production records were maintained.

Appropriate amounts of cadmium chloride (reagent grade) were mixed with the soybean meal just prior to dosing each time. This treated soybean meal portion was layered on top of the grain allotment which was avidly consumed. This allowed a highly uniform exposure to metal every day. Feed and water were continuously sampled to determine the cadmium intake in these animals from control feed and water. The amount of cadmium was 40 mg (for low cadmium group) and 200 mg (for high cadmium level) per day. The animals were divided into groups of four each.

The pigs used in these studies were castrated Ireshire-Yorkshire crossbred males. Thirty weaners of nearly 20–25 kg weight (8–10 weeks of age) were obtained. All animals were held for one week for acclimation with new surroundings. They were kept in groups (initially 4-5 each but later no more than two each in stalls of approximately 1.2 × 2.4 m in dimensions, with a wooden slatted floor. Each stall had swine feeders where a known amount of feed was periodically placed. The amount of feed consumed was recorded by periods. The watering system was automatic and no recording of water consumed was made. All animals were weighed at three-week intervals.

The swine ration was supplemented with known

amounts of cadmium chloride. The weaners were given commercial "grower" ration (18% protein) for first six weeks of experiment. The animals after this period were fed with commercial "finisher" hog ration (14% protein). The cadmium levels were 2 and 10 ppm added to control feed from a premix-feed containing 300 ppm of this metal. Ten animals per group were exposed to the control or cadmium supplemented rations.

The chickens used in these experiments were White Leghorn layers. The animals were nearly 11 months of age when procured and were all producing eggs. These animals were nearly a year old, at the time of starting metal feeding.

The birds were kept in poultry stalls that were approximately 2.5 × 4 m in size and were equipped with feeders, a continuous flow watering system, and laying nests and perches. The feed was offered every day. Periodic records of egg production were kept and eggs and tissues were sampled periodically. The cadmium levels in respective experiments were 2 and 10 ppm added to control feed.

Metal Feeding Schedules and Sampling

Cows were treated with supplemental cadmium for three months after which two animals from each group were sacrificed. The other animals were given control feed for an additional three month period. Milk, feed, and water were periodically sampled for cadmium analyses. At the time of sacrifice, all organs were examined and tissues sampled.

The pigs were given cadmium-treated feed for a period of six months. Selected animals from each group were taken off the treated feed after an initial three months of treatment and given control ration for the rest of the experiment. Two animals from each group were sacrificed at six-week intervals and their tissues sampled for analyses.

Chicken were given cadmium-supplemented feed for 24 weeks. A number of animals were given cadmium only for an initial six weeks duration and were then given control ration for an additional seven weeks to observe the depletion of cadmium from the tissues. Two or three animals from each group were sacrificed after 1, 2, 4, 6, 7, 8, 13, 18, and 24 weeks on treated feed and their tissues were sampled for cadmium analyses.

Detailed necropsy and histopathologic examination of selected tissues were conducted in all three species at the time of sacrifice.

Analytical Procedures

Analyses on milk were performed on a Jarrell-Ash model 810 dual channel AA spectrophotometer, using the graphite tube atomizer for milk samples. Cadmium was measured by using the 2288 nm absorption line with the reference channel monitoring nonspecific absorption at 2265 nm. Milk was digested with HNO_3 in sealed polyethylene bottles. The digestion period was overnight at 70°C. Upon opening the bottle the contents were diluted to 10 ml with HNO_3 and a portion removed and added to an equal volume of 10% (v/v) H_2O_2. The sample digest containing hydrogen peroxide was incubated for 10 min at 80°C and immediately analyzed. Harleco Standard cadmium solution (1000 ppm) was used to prepare working standards for this work.

Analyses of the National Bureau of Standards bovine liver standard using above method gave a mean value of 0.289 ± 0.004 ppm cadmium. The certified cadmium content of that sample was 0.27 ± 0.04 ppm. Recovery of cadmium (0.1 μg added prior to sample digestion) ranged from 90 to 125% with various tissue and milk samples as the background matrix.

All other tissues were ashed at 550°C after initial drying and dissolved in dilute HCl. Cadmium contents were measured by flame atomic absorption spectrophotometry using an appropriate background correction as mentioned above. The standard bovine liver sample (National Bureau of Standards) was frequently used for verification of the method.

Renal and hepatic cadmium binding protein (metallothionein) was estimated in these tissues by the method of Piotrowski et al. (*7*). The details of this procedure and validity of the method used have been described elsewhere (*8*).

Results

The actual concentrations of cadmium in the ration of cattle, swine and chicken, as measured by feed analyses are indicated in Table 1. In the case of cows, although the cadmium was given once daily, the concentrations are based on an average daily intake of total feed (hay, concentrate, and soybean meal) so that even the level in control diet has been adjusted on the basis of cadmium in the various

Table 1. Cadmium concentration in feeds of different species.

Group	Cd, ppm[a]		
	Cows	Swine	Chicken
Control	0.18[b]	0.23	0.32
Low Cd	2.40	2.41	1.88
High Cd	11.29	10.12	13.06

[a] Values in feed averaged from the samples obtained during the experiment.

[b] Based on the amount of cadmium in different feed constituents (grain, soybean meal and hay) and average 18 kg total dietary intake/day.

components of the feed. In other two species, the purpose was to provide an additional supplementation of 2 and 10 ppm of cadmium to the homogeneous mixed feed and the data in Table 1 indicate that this was effectively achieved.

Treatment of cows, pigs, and chickens with the dietary levels of cadmium did not have any effect on the growth and performance of any of the species. In cows, no differences in the amount of milk produced were noticed after additional intake of cadmium. The body weights of these animals remained essentially unchanged throughout the experimentation. In swine, the body weights of the animals treated with cadmium at the highest level were slightly lower than the control group, but in view of the fact that only two animals were carried through that length of the experiment in this group, the effects were not statistically significant (data not shown). In chickens, neither the egg production nor the body weights were influenced by the supplementation of their ration with cadmium. In all three species, the feed consumption was also unchanged in various treatment groups. No treatment-related gross or histopathological changes were observed in any species at the time of sampling.

In dairy cows, daily feeding of an equivalent of 11.3 ppm cadmium in the total ration did not cause accumulation of this metal in either milk or muscle. No accumulation of cadmium in bones was observed. Significant accumulation of this metal was observed, however, in the liver (Fig. 1) and kidney. At the end of the three-month exposure period, the cadmium contents of cow liver and kidney were increased according to the level of cadmium exposure, although statistical significance was noticed only when cadmium was high in diet (i.e., 11.3 ppm). Another important observation was noticed when the animals were given no cadmium for three months after an initial three-month exposure period; the cadmium levels in cow liver and kidney showed no depletion. The Cd levels in these organs were virtually unchanged in low cadmium-exposure groups whereas in animals given the high cadmium treatment, the concentrations of this metal showed a slight increase after the depletion phase (although not statistically significant). This indicates that cadmium is highly persistent in visceral organs, probably in a protein-bound form, and a slight increase in cadmium level even after a three-month period of no exposure may be due to the redistribution of cadmium from other tissues.

Exposure of swine to cadmium-treated ration provided results similar to those in cattle. Feeding a diet containing approximately 10 ppm Cd for a period up to six months did not cause accumulation of this metal in muscle, bone, or brain. However, dose-related increase of Cd concentration occurred in both liver (Fig. 2) and kidney. As early as six

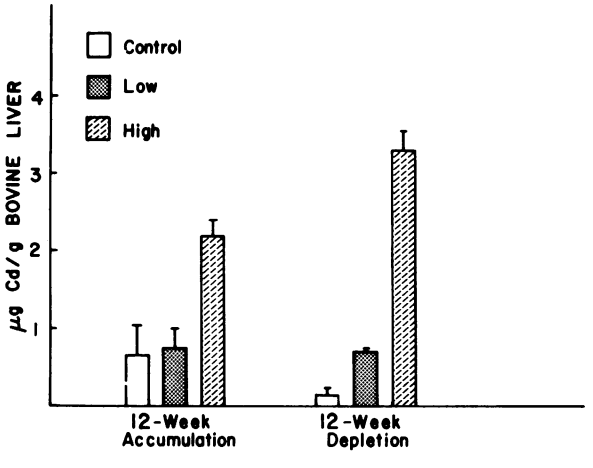

FIGURE 1. Accumulation of cadmium in bovine liver after a period of three months exposure to various dietary levels of the metal and then again three months after the metal supplement had been stopped. The values are means ± SD of two to four samples (ppm fresh weight basis) obtained from two animals at each time interval.

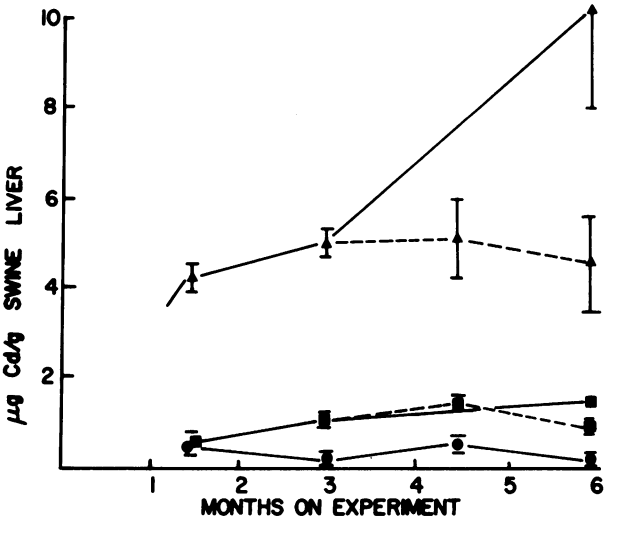

FIGURE 2. Concentration of cadmium in swine liver at different periods of continuous cadmium exposure in diet. The animals were given control or cadmium ration for a period of six months and two animals from each group sacrificed at six-week intervals. The values are mean ± SD (ppm fresh weight basis) of two to four samples in each case and are indicated as (●) control, (■) low cadmium diet, and (▲) high cadmium diet. Points connected with solid lines indicate a continued exposure, whereas a depletion phase (animals exposed to control ration after initial exposure to treated feed) is indicated by broken lines.

weeks after a continuous exposure, swine kidney showed a significant increase in Cd concentration at the low level of Cd in diet (2.4 ppm). Both liver and kidney levels were significantly higher than controls at three months of continuous exposure to a low level of cadmium. At the high level of exposure (10.1 ppm Cd in diet) a drastic and statistically significant increase was seen in both liver and kidney. The levels of cadmium in kidney were nearly 2-4 times higher than those in liver. The increase in liver and kidney cadmium continued through six months of exposure to treated feed (when the experiment terminated) and approached an average of 11 ppm of cadmium in liver and 42 ppm in kidney (on fresh tissue basis) of the animals treated with the high cadmium diet (10.1 ppm). No significant depletion of cadmium was observed in either liver or kidney when the animals were given the control diet (0.3 ppm Cd) for a period up to three months after an initial three month exposure.

In the case of swine kidney, the levels of cadmium were determined separately in medulla and cortical tissue (Table 2). In general, the values for cortex were higher than those for Cd in medullary tissue, particularly as the levels of Cd increased in relation to the level of dietary Cd and duration of exposure. The medullary Cd levels did not show a dose- and time-related increase.

Feeding of Cd in chicken diet (up to 13 ppm) did not cause any increase of Cd levels, either in eggs (Table 3) or in chicken bones. The levels of cadmium were significantly increased in chicken muscle (Table 4), although the slight increase may not be appreciable in terms of human dietary intake for this metal, considering the consumption of poultry meat. The normal chicken muscle had cadmium levels of an average of 0.06 ppm, comparable to the values observed in bovine or swine muscle. In the group of birds fed with 1.9 ppm Cd in diet, the average cadmium level in muscle rose to a value of 0.14 ppm and in chickens exposed to high cadmium diet (13 ppm), the corresponding value was 0.26 ppm after six months of exposure to supplemented feed. The accumulation and persistence of Cd in chicken liver and kidney are shown in Figures 3 and 4.

In all three species of animals, treatment with cadmium caused a dose and time-related increase of cadmium-binding protein (metallothionein, MT). The details of metallothionein levels in these animals have been reported elsewhere (*8*). Significant linear correlations between the metallothionein content and the cadmium concentration were noted both in liver and kidney of all animals except for bovine liver. The increases in the metallothionein levels in kidney cortex were more pronounced than those in liver, and this has been reflected in higher amounts of cadmium accumulation in renal cortex as compared to that in liver.

Figure 5 indicates the relationship of hepatic and renal metallothionein levels. It is apparent that in both organs the contents of this metal-binding protein increased simultaneously, although a relatively

Table 2. Relative cadmium concentrations in kidney medulla and cortex at selected intervals of continuous cadmium feeding in swine.

Group	Tissue (kidney)	Cd concn, μg/g fresh weight[a]		
		3 mo. exposure	6 mo. exposure	3 mo. exposure and 3 mo. depletion
Control	Medulla	0.09 ± 0.03	—	0.08 ± 0.03
	Cortex	0.06 ± 0.00	—	0.15 ± 0.03
Low cadmium	Medulla	1.14 ± 0.70	1.11 ± 0.16	0.76 ± 0.24
	Cortex	6.52 ± 0.82	10.97 ± 0.50	—
High cadmium	Medulla	5.15 ± 2.27	3.08 ± 0.87	2.50
	Cortex	28.12 ± 2.88	42.30 ± 6.99	24.12 ± 0.93

[a] Mean ± SD of 2-6 analyses, except a single analysis where no SD is shown.

Table 3. Cadmium content of egg yolk during and after ingestion of cadmium-treated feed.[a]

Treatment	Cd, μg/g fresh weight (during continuous exposure)				After 7 weeks of depletion after 6 weeks of continuous cadmium exposure
	5 weeks	6 weeks	13 weeks	24 weeks	
Control	0.07 ± 0.00	0.13 ± 0.03	0.10 ± 0.08	—	—
Low cadmium	0.05 ± 0.01	0.09 ± 0.02	0.13 ± 0.01	0.06 ± 0.00	0.14 ± 0.04
High cadmium	0.06 ± 0.01	0.09 ± 0.03	0.15 ± 0.03	0.09 ± 0.02	0.08

[a] The values are ppm in fresh yolk and given as mean ± SD of 2-6 determinations. The values in egg-white (albumin) were on the same order or generally lower than in yolk.

Table 4. Cadmium concentration in chicken muscle given different levels of dietary cadmium for 6 months.[a]

Group	Cadmium in feed, ppm	Cadmium in muscle (fresh weight), ppm
Control	0.32	0.063 ± 0.050
Low	1.88	0.140 ± 0.025[b]
High	13.06	0.263 ± 0.023[b]

[a] Average ± SD of 3–6 samples per group (obtained from 2–3 animals in each group).

[b] Significantly different at $p < .05$ from control values.

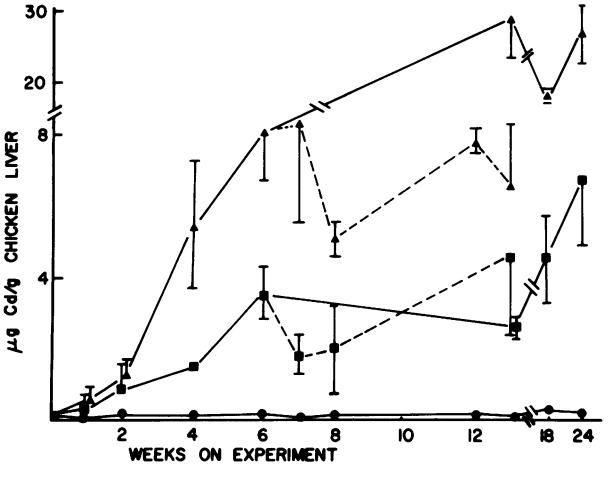

FIGURE 3. Accumulation and persistence of cadmium in chicken liver in animals given various dietary levels of this metal. The values are means ± SD of two to six samples (ppm fresh weight basis) obtained from up to three animals in each group. The animals at various dietary levels are shown as (●) control, (■) low cadmium, and (▲) high cadmium level. The points connected by broken lines indicate the groups that were given control diet after an initial exposure to cadmium-treated diet.

larger increase in renal cortex was seen. When the ratio of cadmium to metallothionein is compared in each organ, it is evident that the synthesis of metallothionein was not to the same extent as the increase of the cadmium content. With increasing cadmium levels, an increasing ratio of cadmium to metallothionein was observed and such change was related in both hepatic tissue and the renal cortex (Fig. 6). The correlation coefficients of parameters in Figures 5 and 6 are indicated in Table 5.

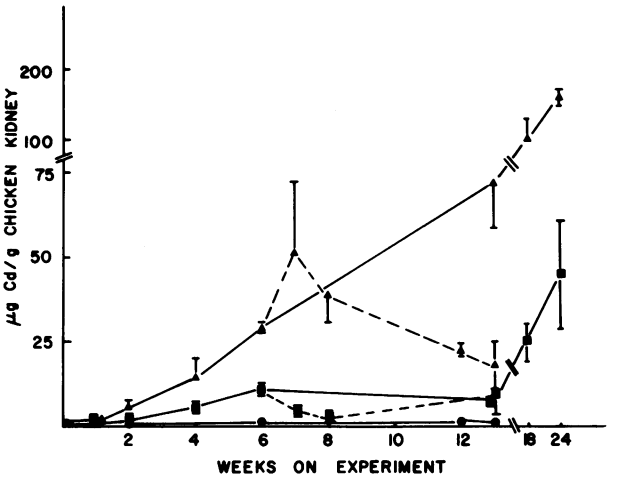

FIGURE 4. Accumulation of cadmium in chicken kidney after the animals were exposed to cadmium-containing diet for a period of up to 24 weeks. All values are from same animals as in Figure 3 and indicate mean ± SD of two to six values (ppm on fresh tissue basis). Some of the groups were treated with cadmium-supplemented ration for an initial six-week period and were then given control ration. Symbols are as for Fig. 3.

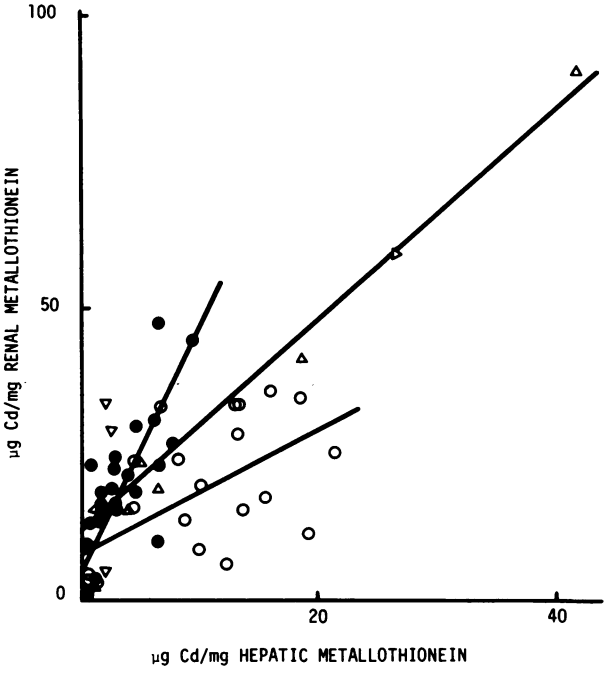

FIGURE 5. Relationship of cadmium and cadmium-binding protein, metallothionein (mg/g wet wt) in liver and kidney cortex (whole kidney of chicken) from animals treated with diets containing different levels of the metal: (△, ▽) cattle, (●) swine, and (○) chicken. The correlation coefficients are shown in Table 5.

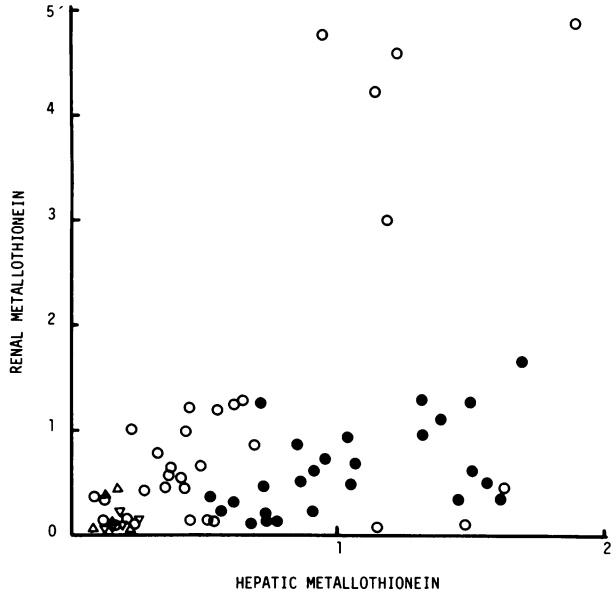

FIGURE 6. Relationship of cadmium-metallothionein ratios in liver and kidney of three food producing animals species: (△, ▽) cattle, (●) swine, and (○) chicken. Although an increase of cadmium caused an induction of metallothionein in both of these organs, the metal/protein ratio increased with increasing level of the metal. This increase was significantly related in different species (correlation coefficients in Table 5). Kidney cortex of cattle and swine, and whole kidney of chicken were analyzed.

Discussion

There was no accumulation of cadmium observed in milk and meat from either cows or swine, or eggs from poultry. Increasing the concentration of cadmium in animal feed up to or more than 10 ppm will cause no appreciable change in the human dietary intake for this metal through the consumption of these organs. Cadmium does not accumulate in bones, and use of bones in chicken feed will have no influence on the cadmium levels of such products. Increasing levels of cadmium were seen, however, in liver and kidneys of all three species and in poultry meat after six months of exposure to cadmium-supplemented ration. The allowable maximum daily human dietary intake of cadmium has been set at 71.4 μg, and it is anticipated that the mean adult intake of cadmium in the U.S. is 72 μg/day (*9*). Any additional cadmium in human food is certain to increase the dietary intake for this metal. It is therefore suggested that the liver and kidney and possibly poultry meat should be avoided in case of a contamination of animal feed with cadmium. Careful monitoring of these products (liver and kidney) is also suggested, particularly if an increase of cadmium level in animal feeds is suspected.

Even though an average value of 0.26 ppm was observed in poultry meat from chickens exposed to high dietary level of cadmium for six months, this value is far less than those reported (*10*) in sea foods (0.79 ppm) and other meats (0.88 ppm). Some of these high values probably occur from contamination of meat through processing. The estimated average intake in man described above has included some of these high values, and on this basis poultry meat does not constitute a hazard as far as the intake of cadmium is concerned.

The contamination of meats and other animal products should be further emphasized. The increased cadmium contents of the tissues of animals and products should be considered in the light of contamination that may be introduced during the processing of foods. As mentioned above high levels of cadmium were found in prepared or processed foods. High levels of cadmium were noticed in sliced bacon, frankfurters, and processed or canned food products (*11*). High cadmium contents of sea food and certain meats (0.79 and 0.88 ppm, respectively) as mentioned above were also well above the cadmium residues of most of the tissues

Table 5. Hepatic and renal cadmium and metallothionein and their correlations in three species of domestic animals.

	Ranges for Cd and MT[a]				Correlation coefficient (r)[b,c]	
	Liver		Kidney			
Species	Cd, μg/g	MT, mg/g	Cd, μg/g	MT, mg/g	Renal Cd/MT vs. hepatic Cd/MT	Renal MT vs. hepatic MT
Cow	0.2- 3.4	0.1-1.6	0.1- 22.1	0.04-0.4	0.9367 ($p < 0.01$)	0.0062 ($p > 0.05$)
Pig	0.1-12.7	0.5-1.7	0.1- 50.1	0.1 -1.7	0.8554 ($p < 0.01$)	0.5281 ($p < 0.01$)
Chicken	0.04-29.8	0.1-1.9	0.1-172.5	0.1 -4.8	0.5860 ($p < 0.01$)	0.6220 ($p < 0.01$)
Overall					0.7433 ($p < 0.01$)	0.4565 ($p < 0.01$)

[a] Concentrations per gram fresh tissue.
[b] Obtained from data shown in Figures 5 and 6.
[c] Values in parentheses indicate levels of significance.

and products of animals treated with cadmium supplemented feed (except for liver and kidneys). It is therefore likely that a major source of contamination of food with cadmium may be processing and storage, rather than the dietary intake of metal by food producing animals. Consumption of organs like liver and kidney should, of course, be avoided if animal feed has been contaminated with cadmium. The daily dietary intake of cadmium in the U. S. is already approaching the maximum allowed tolerance limits (*12*).

Results presented above and also reported previously (*8*) indicate that the accumulation of cadmium in liver and kidney is partly attributable to the increased levels of metallothionein in these tissues. Induction of this cadmium-binding protein was observed in both of these organs related to the cadmium treatment level and the duration of exposure. An increasing ratio of cadmium to metallothionein with increasing cadmium level of the tissue suggest that either the metal-binding protein is being saturated with cadmium at higher levels of this element in the tissues or more of the metal is present in tissues which is not necessarily bound to the metallothionein, or both.

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REFERENCES

1. Lynch, G. P., et al. Physiological responses of calves to cadmium and lead. J. Animal Sci. 42: 410 (1976).
2. U. S. Department of Health, Education and Welfare (1966). Cited by R. C. Dorn, Cadmium, a review. FDA Report 3526-76F, June 1976.
3. Fleischer, M., et al. Environmental impact of cadmium. A review by the panel on hazardous trace substances. Environ. Health Perspect. 7: 253 (1974).
4. Cannon, H. L., and Anderson, B. M. The geochemists involvement with the pollution problem. Geol. Soc. Am. Mem. 123: 155 (1971).
5. Gish, C. D., and Christensen, R. E. Cadmium, nickel, lead and zinc in earthworms from roadside soil. Environ. Sci. Technol. 7: 1060 (1973).
6. Somers, L. E. Chemical composition of municipal sewage sludges and evaluation of their potential use as fertilizers. Cited in: Application of sewage sludge to cropland: appraisal of potential hazards of the heavy metals to plants and animals. U. S. EPA Report 430/9-76-013, 1976.
7. Piotrowski, J. K., Bolanowska, W., and Sapota, A. Evaluation of metallothionein content in animal tissues. Acta Biochem. Pol. 20: 207 (1973).
8. Verma, M. P., Sharma, R. P., and Street, J. C. Hepatic and renal metallothionein (MT) levels in cows, pigs and chickens given cadmium and lead in feed. J. Am. Vet. Res. in press.
9. Joint FAO/WHO Expert Committee on Food Additives, 16th Report, Geneva, 1972.
10. Schroeder, H. A., et al. Essential trace metals in man: zinc relation to environmental cadmium. J. Chronic Dis. 20: 179 (1967).
11. Food and Drug Administration, Bureau of Foods: Compliance Program Evaluation. FY1974 heavy metals in food survey, 1975.
12. Mahaffey, K. R., et al. Heavy metal exposure from foods. Environ. Health Perspect. 12: 63 (1975).